

Depression of the evoked quantal acetylcholine secretion in frog neuromuscular junction by phospholipases A2 from the venom of steppe viper *Vipera ursinii renardi*

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Abstract

© 2018 Russian Academy of Sciences. All rights reserved. In this work we studied the influence of phospholipase A2 vurt toxin and its homologue lacking enzymatic activity Vur-S49 isolated from the venom of steppe viper *Vipera ursinii renardi* on the amplitude-Temporal parameters of spontaneous and evoked endplate currents (EPCs) in the neuromuscular junction of frog *Rana ridibunda*. The experiments performed showed that both vurt toxin and Vur-S49 reduce the EPC quantal content. The amplitude and time course of spontaneous (one-quantal) signals remained unchanged, suggesting that these proteins do not block nicotinic acetylcholine receptors (nAChRs) on the postsynaptic membrane. Depressing effect in the presence of enzymatically inactive Vur-S49 suggested that the decrease in the EPC quantal content under the action of these proteins could not be explained exclusively by phospholipolytic activity manifested by vurt toxin. Relying on our previous data we suggested interaction of the proteins studied with presynaptic $\alpha 7$ nAChRs. Selective antagonist of $\alpha 7$ nicotinic receptors methyllycaconitine (MLA) reduced the EPC quantal content as well. Depressing action of MLA on the evoked secretion of acetylcholine indicates the involvement of $\alpha 7$ nAChRs in regulation of the evoked quantal secretion in the frog neuromuscular junction. However, the effects of vurt toxin and Vur-S49 on the EPC quantal content were unchanged after preliminary incubation of the nerve-muscle preparation with MLA. The data obtained suggest that presynaptic actions of the proteins studied are not directed to $\alpha 7$ nAChRs but could be mediated by interaction with some other synaptic targets.

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Keywords

Endplate current, Neuromuscular junction, Nicotinic acetylcholine receptor, Phospholipase, Viper venom

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